A Weight Reduction and Weight Maintenance Program With Long-Lasting Improvement in Left Ventricular Mass and Blood Pressure

Etsuro Himeno, Kenshi Nishino, Tetsuya Okazaki, Hiroki Nanri, and Masaharu Ikeda

Obesity is a major risk factor for cardiovascular disease and is associated with hypertension and increased left ventricular mass (LVM). Maintenance of reduced weight has been a matter of recent concerns in the treatment of obese subjects. This study was conducted to confirm the effect of the addition of exercise to diet on maintenance of body weight in a weight reduction program. In addition, this study was conducted to estimate whether LVM changes in parallel with a change in body weight during a long-term follow-up after a weight-reduction program.

Twenty-two normotensive (NT) obese subjects and 14 mild hypertensive (HT) obese subjects ranging in age from 22 to 51 years participated in a 12-week supervised weight-reduction program involving mild exercise and a mild hypocaloric diet. After this 12-week intervention, they were advised to maintain their modified lifestyle during a 1-year follow-up period. After the 12-week intervention, the mean reductions in body weight (BW) in the NT and HT groups were 4.1 kg ($P < .0001$) and 5.8 kg ($P < .0001$), respectively. LVM in the NT and HT groups was significantly reduced from 154 g to 136 g ($P < .005$) and from 169 g to 152 g ($P < .002$), respectively. One year after intervention, the mean gains in BW in the NT and HT groups were 2.3 kg (not significant, NS) and 0.4 kg (NS), respectively. The mean gains in LVM in the NT and HT groups were 8 g (NS) and 7 g (NS), respectively. It was also shown that blood pressures in the HT group were significantly decreased after the 12-week intervention and there was no significant change in blood pressure in the HT group 1 year after intervention.

In conclusion, reduced body weight was maintained for 1 year after a 12-week supervised weight-reduction program in both normotensive and mild hypertensive obese subjects. Reduced left ventricular mass was maintained for a long period in both normotensive and mild hypertensive obese subjects and lowered blood pressure was maintained in the mild hypertensive obese subjects. Am J Hypertens 1999;12:682–690 © 1999 American Journal of Hypertension, Ltd.

KEY WORDS: Obesity, mild hypertension, weight maintenance, left ventricular mass, echocardiography.
Obesity is a major risk factor for cardiovascular disease and is associated with hypertension, hyperlipidemia, glucose intolerance, and increased left ventricular mass.\textsuperscript{1–4} Many studies have shown that weight reduction and exercise training in obese subjects can help to improve these risk factors.\textsuperscript{5–8} There are many reports on successful weight-loss programs, but in practical terms weight regain is a serious problem in the treatment of obese subjects. Prevention of obesity naturally depends on long-term maintenance of an isocaloric state and it is widely accepted that the addition of exercise to diet intervention produces more weight loss than does dieting alone. Particularly, exercise appears to be important in maintaining weight loss during follow-up after active intervention.\textsuperscript{9} We thus conducted a 12-week supervised exercise program in which we aimed for successful long-term maintenance of reduced weight after a weight reduction program.

Similarly, left ventricular hypertrophy is a powerful predictor of serious cardiovascular sequelae.\textsuperscript{10} There have been several reports on the reduction in left ventricular mass due to weight loss.\textsuperscript{11–13} MacMahon et al showed that weight reduction in hypertensive obese subjects regressed left ventricular mass, and about 25% of this reduction in left ventricular mass could be explained by the change in body weight.\textsuperscript{11} Alpert et al reported that weight loss by gastroplasty in nonhypertensive extremely obese subjects regressed left ventricular mass.\textsuperscript{12} Our previous report also showed that weight reduction caused by mild exercise and a mild hypocaloric diet in both normotensive and mild hypertensive obese subjects regressed left ventricular mass, and this change in left ventricular mass tended to be associated with the change in body weight.\textsuperscript{13} Thus there have been several reports on the reduction in left ventricular mass due to weight loss, but there have been no follow-up studies regarding the relationship between body weight and left ventricular mass after a weight-reduction program. This study was also conducted to estimate whether a change of left ventricular mass runs parallel with a change in body weight for a long period during a follow-up after a weight-reduction program.

**MATERIALS AND METHODS**

**Study Design** In this study, normotensive and mild hypertensive obese subjects participated in a 12-week supervised weight-reduction program that involved mild exercise and a mild hypocaloric diet. After this 12-week intervention, the subjects were advised to maintain their modified lifestyle during a 1-year follow-up period. We made no intervention with regard to exercise training or food intake during this period. Figure 1 shows the intervention and follow-up schedule. Hemodynamics, biochemical, and constitutional parameters were measured at points (1), (2), and (3).

**Subjects** Although 49 obese subjects initially participated in this program, 13 subjects were excluded. In seven subjects, echocardiograms were technically inadequate, four moved elsewhere, and two started antihypertensive medication during the follow-up period. Eventually, data from 22 (18 men and four women) normotensive obese subjects (NT group) and 14 (13 men and one woman) mild hypertensive obese subjects (HT group) whose systolic blood pressure was between 140 and 160 mm Hg or diastolic blood pressure was between 90 and 100 mm Hg were analyzed in this study. All of the subjects were outpatients. The subjects ranged in age from 22 to 51 years (mean ± SD, 35 ± 9 years). All of the subjects had a body mass index, calculated as body weight (kg)/(body height [m])\textsuperscript{2}, of > 26 kg/m\textsuperscript{2}. None of the subjects had any evidence or history of coronary heart disease, valvular heart disease, congestive heart failure, renal disease, or secondary hypertension. None of the mild hypertensive obese subjects was taking any antihypertensive medications before this study. One subject who had been diagnosed as having diabetes mellitus was included in this study because the fasting blood glucose level was controlled within normal limits by diet. We obtained the informed consent of each of the participants before the program was started.

**Measurements: Physical Characteristics, Hemodynamics, and Plasma Lipids** Body weight (BW), body fat, circumferences of the waist and hips, resting blood pressure, and heart rate were measured three times: at baseline, at the end of the 12-week intervention, and at 1 year after intervention. Body fat was measured with a BFT-2000 (Kett Co, Tokyo, Japan), which measures the absorption of infrared radiation by adipose tissue. The circumferences of the waist and hips were measured at the level of the umbilicus and at the greatest girth at the gluteus, respectively. The waist-hip ratio was calculated as the circumference of the waist divided by the circumference of the hip. Blood pressure was measured after 5 min of rest in the sitting position with a cuff connected to a mercury sphygmomanometer.
meter. Mean blood pressure was calculated as diastolic pressure plus pulse pressure divided by 3. Blood pressure was measured twice and calculated as the mean of the two recordings. Heart rate was also measured at the same time. Total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG) were measured after at least a 12-h fast. Low-density lipoprotein cholesterol (LDL-C) was calculated as TC minus HDL-C minus TG/5.

**Exercise Training** At baseline, all of the subjects performed a cardiopulmonary exercise test with a cycle ergometer (STB-1350, Nihon Koden, Tokyo, Japan) to determine the exercise intensity for exercise training. Exercise testing with expired gas analysis began with 3 min of pedaling at 20 W at 50 rpm as a warm-up, followed by an increase of 30 W every minute in men or an increase of 20 W every minute in women, up to a symptom-limited maximum workload. Heart rate was monitored throughout exercise testing using a 3-lead electrocardiograph. Blood pressure was measured every minute with an automatic indirect manometer (STBP-780B, Colin, Nagoya, Japan). Expired gas analysis was performed with a Sensormedics 4400tc (Sensormedics, Anaheim, CA). The system was calibrated immediately before each measurement. Oxygen uptake, carbon dioxide production, and minute ventilation were measured every 15 sec. The anaerobic threshold (AT) was determined by the V-slope method according to Beaver et al.14 Exercise training threshold (AT) was determined by the V-slope method. Exercise testing with expired gas analysis began with 3 min of pedaling at 20 W at 50 rpm as a warm-up, followed by an increase of 30 W every minute in men or an increase of 20 W every minute in women, up to a symptom-limited maximum workload. Heart rate was monitored throughout exercise testing using a 3-lead electrocardiograph. Blood pressure was measured every minute with an automatic indirect manometer (STBP-780B, Colin, Nagoya, Japan). Expired gas analysis was performed with a Sensormedics 4400tc (Sensormedics, Anaheim, CA). The system was calibrated immediately before each measurement. Oxygen uptake, carbon dioxide production, and minute ventilation were measured every 15 sec. The anaerobic threshold (AT) was determined by the V-slope method according to Beaver et al.14 Exercise training was performed for 1 h three times a week at a heart rate corresponding to AT as determined from the initial exercise test. Mean heart rates corresponding to AT in the NT and HT groups were 114 ± 7 beats/min and 113 ± 11 beats/min, respectively. There was no significant difference between the two groups. Exercise training consisted of pedaling a cycle ergometer (33 subjects) or walking on a treadmill (three subjects) for 30 min, and stretching and walking or jogging for 30 min. To estimate the quantity of exercise training, we established a relative training time, which was calculated as the number of times that the subject performed exercise divided by all of the training sessions scheduled (36 times) multiplied by 100 (%). At baseline and at 1 year after intervention, the physical activity of all of the subjects was evaluated semiquantitatively and graded into four ranks (frequently = 4 points, sometimes = 3 points, rarely = 2 points, sedentary = 1 point) by a self-assessment questionnaire.

**Diet** The standard caloric intake per day for each subject was calculated using the dietary allowances recommended by the Ministry of Welfare in Japan. The target caloric intake was calculated as the standard caloric intake minus 1000 kcal per day. The target caloric intake was 1200 kcal for four subjects, 1400 kcal for three subjects, 1600 kcal for 19 subjects, and 1800 kcal for 10 subjects. Protein, fat, and carbohydrate contributed 12% to 15%, 20% to 25%, and 57% to 68% of the target caloric intake, respectively. Before the intervention, information regarding daily food intake was obtained from all of the subjects by a self-assessment nutritional questionnaire. After intervention and at 1 year after intervention, we obtained information regarding daily food intake using the same questionnaire. Based on this information, we calculated daily salt intake. In addition to the target caloric intake, we established approximately four to eight target items for each subject according to this information. The target items generally included instructions to not eat snacks, to limit alcohol or beverage intake, and to reduce fatty foods, carbohydrates, and so on. To estimate dietary modification, a diet score was calculated to determine whether the subjects had adhered to the target items in the advised diet during the 12-week period. The diet score was calculated from information that each subject provided on a self-assessment questionnaire. For each item, a score of 100 points was given if they followed the advice exactly, 50 points if they followed the advice insufficiently, and 0 points if they did not follow the advice at all. The diet score (points) was calculated as a percentage of the total number of points available.

**Echocardiography** Two-dimensional guided M-mode echocardiography was performed using an Aloka SSD-860 (Tokyo, Japan) equipped with a 3.5-MHz transducer. All echocardiograms were taken in the left lateral position after 5 min of rest. Echocardiograms from the left ventricle were recorded at the level of the tips of the mitral valve leaflets. The left ventricular internal dimension in diastole (LVIDd) and the thicknesses of both the interventricular septum (IVST) and the posterior wall (PWT) were measured using both the American Society of Echocardiography and the Penn convention parameters. Left ventricular internal dimension in systole (LVIDs) was measured at the nadir of septal motion. The strip chart recorder was driven at a paper speed of 50 mm/sec. All measurements were made with a Cardio 500 (Kontron Instruments, Dortmund, Germany). Left ventricular mass (LVM) was calculated according to the formula of Devereux and Reichek:15 LVM (g) = 1.04 [IVST + PWT + LVIDd]3 − (LVIDd)3 − 13.6. All measurements were obtained in at least five cardiac cycles. LVM was corrected by dividing by the subject’s height accounting for Levy et al.16 To assess left ventricular systolic function, left ventricular fractional shortening (%) was calculated as (LVIDd − LVIDs)/LVIDd × 100.

**Statistical Analysis** All statistical analyses were performed using the Stat View 4.02 statistical package. All data are expressed as mean values ± standard devia-
All data in subgroups at baseline were assessed by Student’s group t test. Differences in data between baseline and at the end of the 12-week intervention and between the end of the 12-week intervention and at 1 year after intervention were assessed by a paired Student’s t test. Simple regression was used to evaluate the relationships between left ventricular mass/bdy height (LVM/BH) and both BW and BP. P < .05 was considered statistically significant.

**RESULTS**

Clinical characteristics at baseline in the NT and HT groups are summarized in Table 1. Mean ages in the NT and HT groups were 36 ± 7 and 35 ± 9 years, respectively. There were no significant differences in BW, BMI, or body fat between the two groups. Both the systolic and diastolic blood pressures in the HT group were significantly higher than those in the NT group (P < .0001). LVM in the HT group was greater than that in the NT group, but this difference was not significant. No significant difference was observed in fractional shortening (FS) between the two groups. We calculated a relative training time and a diet score to estimate whether exercise and diet intervention were performed equally in both groups during intervention. There was no significant difference between the mean relative training times in the NT and HT groups (65 ± 22% and 71 ± 14%, respectively). At baseline, there was no significant difference in exercise habits between the two groups (NT v HT, mean ± SD, 2.4 ± 0.6 points v 2.4 ± 0.6 points). However, the HT group exercised more than the NT group at 1 year after intervention (NT v HT, 2.4 ± 0.7 points v 3.1 ± 0.8 points, P < .02). There was no significant difference between the mean diet scores in the NT and HT groups (65 ± 22 points and 69 ± 16 points, respectively). There was no significant difference between the mean salt intake at baseline in the NT and HT groups (12.4 ± 3.0 g and 14.0 ± 4.1 g, respectively). After intervention, the mean salt intake in the NT group was significantly decreased from 12.4 ± 3.0 g to 9.7 ± 2.2 g (P < .001). One year after intervention, the mean salt intake in the NT group was increased from 9.7 ± 2.2 g to 11.3 ± 4.1 g, but this change was not significant. After intervention, the mean salt intake in the HT group was decreased from 14.0 ± 4.1 g to 11.5 ± 2.0 g, but this change was not significant. One year after intervention, the mean salt intake in the HT group was 11.6 ± 2.0 g. The changes in BW, body fat, and the waist-hip ratio from baseline to 1 year after intervention in both groups are shown in Table 2. After the 12-week intervention, the mean reductions in BW, body fat, and the waist-hip ratio in the NT group were 4.1 kg (P < .0001), 2.4% (P < .0001), and 0.03 (P < .0001), respectively, whereas these values in the HT group were 5.8 kg (P < .0001), 4% (P < .0001), and 0.04 (P < .0005), respectively. One year after intervention, the mean gains in BW, body fat, and the waist-hip ratio in the NT group were 2.3 kg (NS), 2.5% (P < .0001), and 0.02 (P < .05), respectively. In the HT group, these values were 0.4 kg (NS), 0.8% (NS), and 0.01 (NS), respectively. The changes in hemodynamic parameters from baseline to 1 year after intervention are also shown in Table 2. After the 12-week intervention, systolic blood pressure was significantly decreased by 5 mm Hg (P < .02) in the NT group. Diastolic and mean blood pressures did not change significantly. In the HT group, systolic, diastolic, and mean blood pressures were significantly decreased by 13 mm Hg, 9 mm Hg, and 12 mm Hg, respectively (P < .0001). One year after intervention, systolic, diastolic, and mean blood pressures in both the NT and HT groups did not change significantly. Heart rate in both the NT and HT groups was unchanged from baseline to 1 year after intervention.

The changes in echocardiographic parameters from baseline to 1 year after intervention in both groups are shown in Table 3. After the 12-week intervention, PWT and IVST in the HT group were significantly decreased from 0.99 ± 0.19 cm to 0.84 ± 0.11 cm (P < .02) and from 0.91 ± 0.13 cm to 0.83 ± 0.10 cm (P < .05), respectively. IVST in the NT group was significantly decreased from 0.86 ± 0.20 cm to 0.79 ± 0.15 cm (P < .05). LVM in the NT and HT groups was significantly reduced from 154 ± 33 g to 136 ± 31 g (P < .005) and from 169 ± 31 g to 152 ± 24 g (P < .002), respectively. LVM/BH in both groups was also significantly reduced after intervention. FS in the HT group was significantly increased from 37% ± 5% to 40% ± 14%.
TABLE 2. CHANGES IN BODY COMPOSITION, HEMODYNAMIC PARAMETERS, AND SERUM LIPIDS FROM BASELINE TO 1 YEAR AFTER INTERVENTION IN THE NT AND HT GROUPS

<table>
<thead>
<tr>
<th></th>
<th>NT Group (n = 22) Before</th>
<th>NT Group (n = 22) After</th>
<th>NT Group (n = 22) 1 Year After</th>
<th>HT Group (n = 14) Before</th>
<th>HT Group (n = 14) After</th>
<th>HT Group (n = 14) 1 Year After</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight (kg)</td>
<td>79.8 ± 11.1</td>
<td>75.7 ± 10.8*</td>
<td>78.0 ± 12.2</td>
<td>86.7 ± 14.6</td>
<td>80.9 ± 14.9*</td>
<td>81.3 ± 16.5</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>28.6 ± 3.9</td>
<td>26.2 ± 3.8</td>
<td>28.7 ± 4.4†</td>
<td>29.1 ± 3.2</td>
<td>25.1 ± 3.9*</td>
<td>26.3 ± 5.5</td>
</tr>
<tr>
<td>Waist-hip ratio</td>
<td>0.93 ± 0.04</td>
<td>0.90 ± 0.04*</td>
<td>0.92 ± 0.03‡</td>
<td>0.93 ± 0.07</td>
<td>0.89 ± 0.06§</td>
<td>0.90 ± 0.06</td>
</tr>
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<td>SBP (mm Hg)</td>
<td>128 ± 7</td>
<td>123 ± 8§</td>
<td>124 ± 8</td>
<td>144 ± 9</td>
<td>131 ± 11*</td>
<td>128 ± 10</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>81 ± 6</td>
<td>81 ± 7</td>
<td>81 ± 8</td>
<td>94 ± 5</td>
<td>83 ± 10*</td>
<td>88 ± 9</td>
</tr>
<tr>
<td>MBP (mm Hg)</td>
<td>97 ± 5</td>
<td>95 ± 7</td>
<td>95 ± 7</td>
<td>111 ± 4</td>
<td>99 ± 9</td>
<td>101 ± 9</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>72 ± 10</td>
<td>73 ± 11</td>
<td>74 ± 10</td>
<td>77 ± 10</td>
<td>76 ± 8</td>
<td>76 ± 10</td>
</tr>
<tr>
<td>TC (mg/dL)</td>
<td>205 ± 35</td>
<td>198 ± 35</td>
<td>202 ± 39</td>
<td>209 ± 34</td>
<td>201 ± 36</td>
<td>213 ± 42</td>
</tr>
<tr>
<td>HDL-C (mg/dL)</td>
<td>42 ± 6</td>
<td>45 ± 7*</td>
<td>47 ± 8</td>
<td>46 ± 17</td>
<td>49 ± 19</td>
<td>56 ± 20‡</td>
</tr>
<tr>
<td>TC/HDL-C</td>
<td>5.0 ± 12</td>
<td>4.4 ± 0.8**</td>
<td>4.4 ± 0.9</td>
<td>4.9 ± 1.4</td>
<td>4.4 ± 1.3†</td>
<td>4.2 ± 1.4</td>
</tr>
<tr>
<td>LDL-C (mg/dL)</td>
<td>129 ± 32</td>
<td>126 ± 32</td>
<td>127 ± 30</td>
<td>128 ± 28</td>
<td>123 ± 31</td>
<td>136 ± 39</td>
</tr>
<tr>
<td>TG (mg/dL)</td>
<td>172 ± 89</td>
<td>135 ± 64‡</td>
<td>142 ± 92</td>
<td>170 ± 75</td>
<td>140 ± 89§</td>
<td>105 ± 46</td>
</tr>
</tbody>
</table>

Values are mean ± SD.

NT group, normotensive group; HT group, mild hypertensive group; SBP, systolic blood pressure; DBP, diastolic blood pressure; MBP, mean blood pressure; HR, heart rate; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TG, triglyceride.

* P < .0001 (before v after); † P < .0001 (after v 1 year after); ‡ P < .01 (after v 1 year after); § P < .0005 (before v after); ¶ P < .05 (before v after); ** P < .01; †† P < .02 (before v after).

3% (P < .05). One year after intervention, LVIDd in the NT group was significantly increased from 4.90 ± 0.44 cm to 5.16 ± 0.51 cm (P < .01). Mean gains in LVM in the NT and HT groups were 8 g and 7 g, respectively. The relationships between LVM/BH and BW in the NT and HT groups are shown in Figure 2. The coefficient of correlation between LVM/BH and BW in the NT group was 0.352 (P < .01). However, there was no significant correlation between LVM/BH and BW in the HT group. The changes in plasma lipids from baseline to 1 year after intervention are shown in Table 2. After the 12-week intervention, TC/HDL-C and TG in the NT group were significantly decreased from 5.0 ± 1.2 mg/dL to 4.4 ± 0.8 mg/dL (P < .01) and from 172 ± 89 mg/dL to 135 ± 64 mg/dL (P < .02), respectively. HDL-C in the NT group was significantly increased from 42 ± 6 mg/dL to 45 ± 7 mg/dL (P < .01). In the HT group, mean reductions in TC/HDL-C and TG were 0.5 (P < .05) and 30 mg/dL (P < .05), respectively. One year after intervention, HDL-C in the HT group was significantly increased from 49 ± 19

TABLE 3. CHANGES IN ECHOCARDIOGRAPHIC PARAMETERS FROM BASELINE TO 1 YEAR AFTER INTERVENTION IN THE NT AND HT GROUPS

<table>
<thead>
<tr>
<th></th>
<th>NT Group (n = 22) Before</th>
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<th>NT Group (n = 22) 1 Year After</th>
<th>HT Group (n = 14) Before</th>
<th>HT Group (n = 14) After</th>
<th>HT Group (n = 14) 1 Year After</th>
</tr>
</thead>
<tbody>
<tr>
<td>PWT (cm)</td>
<td>0.89 ± 0.19</td>
<td>0.85 ± 0.18</td>
<td>0.82 ± 0.18</td>
<td>0.99 ± 0.19</td>
<td>0.84 ± 0.11*</td>
<td>0.88 ± 0.11</td>
</tr>
<tr>
<td>LVIDd (cm)</td>
<td>4.91 ± 0.44</td>
<td>4.90 ± 0.44</td>
<td>5.16 ± 0.51†</td>
<td>4.88 ± 0.41</td>
<td>5.13 ± 0.37</td>
<td>4.96 ± 0.42</td>
</tr>
<tr>
<td>IVST (cm)</td>
<td>0.86 ± 0.20</td>
<td>0.79 ± 0.15‡</td>
<td>0.79 ± 0.14</td>
<td>0.91 ± 0.13</td>
<td>0.83 ± 0.10‡</td>
<td>0.89 ± 0.13</td>
</tr>
<tr>
<td>LVM (g)</td>
<td>154 ± 33</td>
<td>136 ± 31§</td>
<td>144 ± 26</td>
<td>169 ± 31</td>
<td>152 ± 24§</td>
<td>159 ± 29</td>
</tr>
<tr>
<td>LVM/BH (g/m)</td>
<td>93 ± 18</td>
<td>82 ± 18**</td>
<td>87 ± 16</td>
<td>101 ± 18</td>
<td>91 ± 14¶</td>
<td>95 ± 17</td>
</tr>
<tr>
<td>FS (%)</td>
<td>37 ± 6</td>
<td>39 ± 6</td>
<td>39 ± 5</td>
<td>37 ± 5</td>
<td>40 ± 3‡</td>
<td>39 ± 5</td>
</tr>
</tbody>
</table>

Values are mean ± SD.

NT group, normotensive group; HT group, mild hypertensive group; PWT, posterior wall thickness; LVIDd, left ventricular internal dimension in diastole; IVST, interventricular septal thickness; LVM, left ventricular mass; LVM/BH, left ventricular mass/body height; FS, fractional shortening.

* P < .02; † P < .01 (after v 1 year after); ‡ P < .05 (before v after); § P < .005; ¶ P < .01; ** P < .002.
mg/dL to 56 ± 20 mg/dL (P < 0.05). The mean reduction in TG in the HT group was 25 mg/dL, but this change was not significant. Plasma lipids in the NT group did not change significantly.

DISCUSSION

Obesity is not only a serious health hazard for metabolic disorders such as diabetes mellitus and dyslipidemia, but is also a major risk factor for cardiovascular disease and is associated with hypertension and increased left ventricular mass. Hence, many attempts to reduce body weight have been made, but most subjects fail to maintain reduced weight, even if successful with weight loss.9 Weight cycling—weight loss followed by weight regain—has been reported to cause possible adverse effects on health.17 Therefore, weight regain is an important problem in the treatment of obese subjects. Prevention of obesity naturally depends on long-term maintenance of an isocaloric state. It is widely accepted that the addition of exercise to diet intervention produces more weight loss than does dieting alone.9 In particular, exercise appears to be important in maintaining weight loss during follow-up after active intervention. Supervised exercise combined with dietary education has been reported to retard the rate of weight gain in obese high school boys.18 We conducted a 3-month supervised exercise program to successfully maintain reduced body weight long-term after a weight reduction program.

In the present study, NT and HT obese subjects participated in a 12-week supervised weight-reduction program involving mild exercise and a mild hypocaloric diet. After this 12-week intervention, they were advised to maintain their modified lifestyle during a 1-year follow-up period. One year after intervention, weight regain in both the NT and HT groups was not statistically significant. However, weight regain in the HT group was less than that in the NT group. The precise reason for this difference is not clear, but may be related to our finding that hypertensive obese subjects seemed to have greater physical activity than normotensive obese subjects after the weight-reduction program in the present study. At baseline and at 1 year after intervention, the physical activity of all of the subjects was evaluated by a questionnaire. There was no significant difference in physical activity between the two groups at baseline. However, physical activity in the HT group was significantly higher than that in the NT group during the follow-up period. In addition, HDL-C was significantly increased whereas TG was decreased from postintervention to 1 year after intervention in the HT group. Increased HDL-C levels and decreased TG levels may be considered as an indicator of maintained physical activity or as a reflection of protocol adherence, as previously reported.9,19,20 Our results showed that body weight in the HT group was maintained from postintervention to 1 year after intervention. This seems to be consistent with a report that continuing exercise contributed to the maintenance of reduced weight.21

FIGURE 2. The relationships between LVM/BH and BW in the NT and HT groups. The coefficient of correlation between LVM/BH and BW was 0.352 (P < .01) in the NT group (a). The coefficient of correlation between LVM/BH and BW was 0.114 in the HT group (b). BW, body weight; LVM/BH, left ventricular mass/body height; NT group, normotensive group; HT group, mild hypertensive group.
Left ventricular hypertrophy (LVH) is a major risk indicator of cardiovascular disease. Body weight, blood pressure, stroke volume, sodium intake, physical activity, and hematocrit are considered to be independent predictors of left ventricular mass (LVM). In obese subjects, body mass index and insulin resistance
have been reported to be important determinants of LVH.\textsuperscript{23,24} MacMahon et al\textsuperscript{11} and Ferrara et al\textsuperscript{25} reported that weight reduction in hypertensive obese subjects reduced LVM and they considered that the reduction in LVM was mainly the result of the reduction in BW.

We have shown that weight reduction caused by mild exercise and a mild hypocaloric diet regressed LVM in normotensive and mild hypertensive obese subjects regardless of blood pressure level.\textsuperscript{13} The mechanisms of the reduction in the LVM that accompanied weight loss are still not clear. As noted, weight regained after a weight-reduction program is a matter of concern in the treatment of obese subjects. Whether or not fluctuations in body weight increase the mortality rate associated with cardiovascular disease is controversial.\textsuperscript{26,27} So far, there have been no follow-up studies regarding the relationship between body weight and left ventricular mass after a weight-reduction program. In this study, we investigated whether left ventricular mass changes in parallel with a change in body weight during a long-term follow-up after a weight-reduction program. In the normotensive and mild hypertensive obese groups, LVM as well as BW did not significantly increase from the termination of a supervised intervention to 1 year after the intervention. However, among echocardiographic parameters, left ventricular internal dimension in diastole was significantly increased in the normotensive group with a slight, but not significant, increase in body weight. This result seems to be consistent with Messerli’s report in which he suggested that normotensive obese subjects predominantly produced eccentric LVH.\textsuperscript{28}

In the NT group, LVM/BH was significantly correlated with BW. On the other hand, LVM/BH in the HT group was significantly correlated with systolic, diastolic, and mean blood pressures. Taken together, these results demonstrate that the mechanisms of the change in LVM in the NT and HT groups may be different, although sample sizes were small. The change in LVM in the NT group may be associated with a change in BW whereas that in the HT group may be largely attributable to a change in blood pressure. However, even in the HT group, the change in BW contributed to the change in LVM to some extent, as it is well known that weight reduction in hypertensive obese subjects decreases blood pressure.\textsuperscript{9} Further studies are required to clarify whether the change in LVM is influenced by significant weight regain and to elucidate the precise mechanism.

The present study also showed that systolic, diastolic, and mean blood pressures in the HT group were decreased by a weight-reduction program and were maintained from postintervention to 1 year after intervention. Previous studies have reported that either exercise training or sodium restriction decreased blood pressure.\textsuperscript{29,30} To clarify the effect of the change in salt intake on the blood pressure level during the study, we calculated daily salt intake by a self-assessment nutritional questionnaire. There was no change in mean salt intake between postintervention and 1 year after intervention. Hence, continued exercise training and the limitation of salt intake during the follow-up period may have contributed to maintaining both blood pressure and body weight.

In conclusion, reduced body weight was maintained for 1 year after a weight-reduction program in both normotensive and mild hypertensive obese subjects during observational intervention. In this case, reduced left ventricular mass was maintained for a long period in both the normotensive and mild hypertensive obese subjects, and blood pressure was maintained normally in the mild hypertensive obese subjects.

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**REFERENCES**

11. MacMahon SW, Wilcken DEL, Macdonald GJ: The ef-


